ium®) is generally regarded as the drug of choice.

Undiluted Valium is slowly administered intravenously at 0.3 mg per kg of body weight up to but not exceeding 10 mg over a 2 minute period. If convulsions are not stopped, the same dose of Valium can be repeated in 20 to 30 minutes. Respiratory arrest has been reported during the fourth administration of Valium, and the drug should be administered slowly and in circumstances where resuscitative emergencies can be reliably handled. If the status epilepticus is still not stopped an anesthesiologist should be consulted to anesthetize the patient.

It cannot be overemphasized that meticulous attention must be paid to the oxygen supply, fluid and electrolytes, and the medications and dosages given must be accurately recorded. Once the status epilepticus is controlled, maintenance doses of anticonvulsants must be assured to avoid recurrence of the seizure activity.

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New Perspectives in the Sudden Infant Death Syndrome

A SEEMINGLY HEALTHY INFANT is put to sleep and found dead in bed. Asphyxiation? Aspiration? Child abuse or neglect? None of these. Rather, the infant is the victim of the sudden infant death syndrome (SIDS, crib death, cot death). Typically, a SIDS episode occurs during sleep or following feeding in infants 1 to 6 months old with an associated upper respiratory infection in the winter months. Premature infants and infants from lower socio-economic strata are particularly at risk. With an incidence of 1 in 10,000 births (8,000 deaths yearly in the United States), SIDS is the second most common cause of death in infancy past the first week of life.

Greater occurrence in twins, in family members, in males and in infants with Type B blood, and a reduced incidence in certain geographic areas such as Israel and parts of Scandinavia, suggest a familial or genetic component.

How sudden is the sudden infant death syndrome? Chronicity is suggested from pathological studies which show that in more than half the victims evidence is seen of chronic alveolar hypoventilation and hypoxemia. Autopsy findings include hypertrophied pulmonary arteriolar muscles, an increased ratio of brown to white fat cells, localized right ventricular cardiac hypertrophy, hypoplasia of the carotid bodies and adrenal medulla, and retarded myelinization and astroglial proliferation in medullary centers of the brain stem.

Human investigation focuses on high-risk target infants: (1) siblings of victims, (2) infants with excessive spontaneous apneic episodes during sleep and (3) "near-miss" victims (resuscitated infants accidentally found cyanotic or white without respiration by caretakers).

Pulmonary studies during sleep define several types of spontaneous apnea: *central apnea,* in which air flow at the mouth and nares ceases concomitantly with cessation of diaphragmatic excursion; *obstructive* or *upper airway apnea,* in which air flow at the mouth and nares ceases despite increasing diaphragmatic efforts, and *mixed apnea,* in which central apnea gives way to obstructive apnea. The obstructive and mixed apneas seem particularly hazardous for a SIDS event because of frequent association with cardiac arrhythmias, including runs of bradycardia and ventricular tachycardia. Although the specific location of the obstruction has not been found, functional obstructions are likely to occur at the level of the larynx. A rich and highly specific anatomical nerve supply makes this area particularly vulnerable for reflexive constriction. Spontaneous cardiac arrhythmias without associated apneas also have been observed. Consequently, SIDS victims may represent neonatal precursors of the "prolonged QT interval syndrome" of adults. Finally, several biochemical, neurohumoral and metabolic deficiencies (immaturities) have been observed. Abnormalities in gluconeogenesis (phosphoenolpyruvate carboxykinase deficiency), dopamine synthesis (decarboxylase deficiency) and magnesium metabolism have been especially implicated.

It is to be hoped that in the not too distant future the pathophysiology and causes of SIDS will be elucidated definitively. What can be done now? Repeatedly, it has been shown that, without support, surviving families are particularly vulnerable to marital discord and psychopathological symp-

toms in individual members. Surviving siblings are especially neglected. Community and social recrimination, personal guilt and feelings of inadequacy, concerns about future family planning and ascribing the role of the victim to a surviving sibling are some of the many problems that develop. Efforts directed toward professional and general public education, and psychological support services for families of victims are required. Parental self-help groups and professional counseling should be available.

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Vitamin E Deficiency in Premature Infants: Interaction with Iron

VITAMIN E DEFICIENCY and iron deficiency can both cause anemia in premature infants but by different mechanisms and at different ages. Vitamin E acts to protect the red cell membranes against oxidative breakdown, and a lack of the vitamin results in anemia due to accelerated destruction of red blood cells. A premature infant may become deficient in vitamin E shortly after birth. The basis for this deficiency is the failure to absorb adequate amounts of the vitamin rather than a dietary lack. By 2 to 3 months of age, or at about the time that a premature infant would normally have been born, absorption of the vitamin becomes normal and the anemia is corrected spontaneously, without a change in diet. In contrast to the early development of vitamin E deficiency, iron deficiency anemia is rarely evident before 4 months of age, after tissue iron stores become exhausted. It is due to a lack of sufficient dietary iron to meet the needs of rapid growth, and it results in a decreased net production of hemoglobin, the major iron-containing protein in the body.

Although iron administration is intended to prevent anemia, large doses (8 mg per kg of body weight) given to preterm infants under 3 months of age may actually accentuate the fall in hemoglobin concentration that normally occurs after birth. In such large doses iron is believed to act as a cofactor in the oxidative breakdown of the red cell membrane which at this age is not well protected by the antioxidant action of vitamin E.

There are two ways to reduce the risk of nutritional anemia in premature infants. The first is to use a new water soluble form of vitamin E, alpha-tocopherol polyethylene glycol-1000 succinate (TPGS), during the first three months of life. This form of the vitamin is adequately absorbed even by small premature infants and is already in use in some commercial multivitamin preparations. The second is to avoid maintenance doses of iron above the 2 mg per kg of body weight per day recommended by the Committee on Nutrition of the Academy of Pediatrics. Indeed, it is acceptable to delay administration of supplemental iron entirely until after 2 or 3 months of age since iron stores are rarely depleted before this time.

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Ampicillin Resistant Hemophilus Influenzae —*A New Dilemma*

BEFORE 1973, many reports of therapeutic failures with ampicillin given during treatment of infections due to Hemophilus influenzae, type B were related to inadequate ampicillin administration or persistent foci of occult infections. However, during the latter part of 1973, strains of H. influenzae resistant to ampicillin were involved in a variety of infections including otitis media, acute epiglottitis, meningitis and septicemia. Subsequently, these resistant strains have been shown to produce a constitutive and cell bound beta-lactamase which rapidly destroys many penicillins and cephalosporins, particularly penicillin G and ampicillin. Minimal inhibitory concentrations for ampicillin among these resistant strains are as high as 50 to 200 micrograms (μg) of ampicillin per ml. Since the beta-lactamase produced by these strains of H. influenzae are similar to those produced by Klebsiella, it is specu-